

# **INTRAPARTUM AMNIOINFUSION FOR MECONIUM STAINED AMNIOTIC FLUID**

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University**

**Chennai, Tamilnadu.**

## **CERTIFICATE**

This is to certify that this dissertation titled **“INTRAPARTUM AMNIOINFUSION FOR MECONIUM STAINED AMNIOTIC FLUID”** submitted by **Dr.J.BEENA** to the faculty of Obstetrics & Gynaecology, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch II (Obstetrics & Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.

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## **DECLARATION**

I, **Dr. J.BEENA** solemnly declare that the dissertation titled **“INTRAPARTUM AMNIOINFUSION FOR MECONIUM STAINED AMNIOTIC FLUID”** has been prepared by me.

This is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of MD Degree Branch II (Obstetrics & Gynaecology) to be held in March 2008.

It was not submitted to the award of any degree/ diploma to any University either in part or in full form previously.

Place : Madurai

Date :

**Dr.J.BEENA**

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## **INTRODUCTION**

The presence of meconium in the amniotic fluid is relatively common. It remains a concern for the obstetrician because meconium passage is associated with increased perinatal morbidity and mortality and its association with meconium aspiration syndrome and its sequelae. Infants born through meconium stained amniotic fluid are more likely to be depressed at birth and require resuscitation and neonatal intensive care.

The significance of presence of meconium in labour is controversial. Meconium aspiration can occur in utero, during the process of birth or after birth. The persistence of meconium aspiration syndrome despite suctioning at birth may be related to intrauterine aspiration.

In the past meconium stained amniotic fluid has been associated with fetal distress. Passage of meconium has been a marker of antepartum or intrapartum asphyxia.

But now invitro passage of meconium is considered a physiological consequence of normal gastrointestinal tract maturation, and this event alone might not indicate fetal distress.

The overall frequency of meconium stained amniotic fluid varies between 10% to 15%. If this meconium stained fluid is aspirated by the

fetus before or during birth, meconium can obstruct the airways, cause inflammation, interfere with surfactant function and cause respiratory difficulties resulting in meconium aspiration syndrome (MAS). The syndrome is characterized by the development of respiratory distress in an infant born through meconium stained amniotic fluid whose symptoms cannot otherwise be explained.

Meconium aspiration syndrome occurs in 5-10% of infants born through meconium stained amniotic fluid and in 10-40% of newborns with meconium below vocal cords. It is a life threatening emergency in term babies. Despite intensive management it is associated with increased mortality of about 10% -40%. It is a leading cause of respiratory distress in the new born.

Infants with severe meconium aspiration syndrome develop pulmonary hypertension and cardiopulmonary failure requiring extracorporeal membrane oxygen (EMCO). Those who survive may demonstrate residual neurological deficits. Therefore there is great interest in developing strategies to prevent meconium aspiration syndrome. Initial attempts were aimed at neonatal management, later a perinatal approach to the problem was recommended including suctioning the oropharynx at birth in infants with meconium stained amniotic fluid. Since the presence of thick meconium was associated with poor outcome

in infant, amnioinfusion was suggested. Amnioinfusion, the instillation of normal saline into the uterine cavity during labour has been proposed as a method

- a) To reduce the meconium concentration by diluting the meconium on the trachea and thus decreasing the potential of developing obstruction of airways and development of meconium aspiration syndrome.
- b) It reduces cord compression in cases of oligohydramnios for which thick meconium is a marker and therefore fetal gasping and prevents inutero aspiration.



## **AIM OF THE STUDY**

1. To evaluate intrapartum transcervical amnioinfusion as a therapeutic intervention in thick meconium stained amniotic fluid.
  - a. To reduce the incidence of caesarean section.
  - b. To prevent meconium aspiration syndrome.
  - c. To reduce perinatal morbidity and mortality in cases of meconium stained amniotic fluid.

## **REVIEW OF LITERATURE**

During the 1950s and 1960s it was thought that meconium stained amniotic fluid was marker of fetal compromise and efforts were made to deliver the foetus either before or as soon as meconium appeared.

Amnioscopy was introduced, for post dated pregnancies and Saldana and associates showed no benefit from amnioscopy and delivery.

Because meconium passage is more common with advanced gestation age, many studies (Cole and associates) have investigated the effect of inducing labour at an earlier gestation between 39-40wk. But no effect on perinatal mortality or respiratory disease was seen.

Later it was widely believed that meconium aspiration developed by inhalation of meconium at delivery when the infant took, its first breath. To prevent this aggressive policies of oropharyngeal suction, endotracheal intubation, cricoid pressure and splitting of thorax were proposed which had little effect on aspiration.

Gabbe and Co-workers (1976) showed in monkeys that removal of amniotic fluid produced variable decelerations and that replenishment of fluid with saline relieved the deceleration.

Miyazaki and Taylor (1983) used amnioinfusion for variable or prolonged decelerations due to cord entrapment. Fetal heart rate pattern in half of the women studied improved.

Miyazaki and Nevarez (1985) found that women treated with amnioinfusion for cord compression pattern, less often required caesarean delivery for fetal distress.

Intrapartum amnioinfusion was initially proposed by Wenstrom and Parsons as a way of diluting the meconium to decrease the incidence of meconium aspiration syndrome. They showed a significant reduction in meconium below cords.

Sadavosky (1989) found that amnioinfusion effectively decreases the frequency of thick meconium, frequency of neonatal acidemia, and frequency of meconium below vocal cords and meconium aspiration syndrome.

Wenstrom and Parson 1989 reported that amnioinfusion decreases meconium below vocal cords, low one minute apgar scores and lower operative delivery rate.

Marci et al (1992) reported significant reduction in fetal distress, significant reduction in caesarean section for fetal distress and decreased incidence of meconium aspiration and meconium aspiration syndrome.

Eriksen et al (1994) reported decreased incidence of meconium below vocal cords and meconium aspiration syndrome.

Cialone et al (1994) reported decreased incidence of meconium below cords, decreased fetal acidemia and meconium aspiration syndrome.

Demeenus et al (1997) reported significant decrease in caesarean sections.

Hofmeyr et al (CRAMP-1) (in South Africa). The Collaborative Randomised Amnioinfusion for Meconium Project study 1998 did not show a significant reduction in caesarean section or meconium aspiration syndrome. However when they pooled their data with Zimbabwe (CRAMP 2) study they demonstrated a significant effect of amnioinfusion in reducing caesarean section and meconium aspiration syndrome.

Puertas et al (1999) reported reduced caesarean section for fetal distress and decreased incidence of meconium below vocal cords.

Pierce et al (2000) in a meta analysis of prospective trials reported that amnioinfusion improves the neonatal outcome and lowers the rate of caesarean delivery and does not increase the rate of postpartum endometritis.

Hofmeyr (2002) in Cochrane Data base review, a systematic review of randomized trials, demonstrated that amnioinfusion reduced the rates of meconium aspiration syndrome and caesarean delivery.

Rathore et al (2002) reported decreased caesarean section rate, decreased incidence of meconium at vocal cords and improved neonatal outcomes.

Shah (2004) reported amnioinfusion as the ideal method of preventing fetal distress due to meconium stained amniotic fluid and reported decreased incidence of meconium aspiration syndrome and perinatal mortality and morbidity.

Sood et al (2004) reported that amnioinfusion decreased the incidence of caesarean section, meconium aspiration and hospital stay.

There are conflicting reports on the results of prophylactic amnioinfusion. Two studies question the routine use of amnioinfusion.

Studies by Spong et al and Usha et al do not indicate any definite improvement with amnioinfusion for meconium stained amniotic fluid.

Spong (1994) and colleagues concluded that there was no difference in outcome between the amnioinfusion group and standard group and suggested that benefit of amnioinfusion is the result of alleviation of variable heart rate deceleration.

Similarly Usha et (1995) reported no difference in meconium aspiration syndrome with amnio infusion.

Hofmeyr Y et al (2007) in a systematic review of randomized controlled trial reported that in clinical settings with standard peripartum care amnioinfusion does not reduce meconium aspiration syndrome, but in setting with limited peripartum surveillance amnioinfusion reduced the risk of meconium aspiration syndrome.

However in a recent study Das et al (2007) observed significant decrease of caesarean deliveries, meconium aspiration syndrome and perinatal deaths.

## **AMNIO INFUSION – GUIDELINES**

### **Definition**

It is the artificial instillation of a sterile solution of normal saline or ringer lactate into the uterine cavity usually performed transcervically during intrapartum period. It is a beneficial intervention in meconium stained amniotic fluid. This procedure is effective and easy to perform and safe.

## **INDICATIONS**

### **Therapeutic**

#### *A. Antepartum*

1. Treatment or prevention of complications caused by oligohydramnios in preterm pregnancies both diagnostic and therapeutic to prevent pulmonary hypoplasia.
2. In pregnancies complicated by preterm PROM, serial amnioinfusion results in significant prolongation of pregnancy and better neonatal outcome.

#### *B. Intrapartum*

1. Intrapartum transcervical amnioinfusion for prevention of meconium aspiration syndrome caused by meconium stained, amniotic fluid.
2. Reduction of variable fetal heart rate deceleration during labour caused by umbilical cord compression.

### **Diagnostic**

In oligohydramnios amnioinfusion can be used for improving ultrasound imaging.

## **Contraindications**

1. Active maternal genital herpes infection
2. Diminished FHR variability or reactivity
3. Fetal scalp PH <7.2
4. Late decelerations in fetal heart rate
5. Placenta praevia
6. Placental abruption
7. Chorioamnionitis
8. Fetal malpresentation

## **Relative contraindications**

1. Fetal anomalies
2. Impending delivery
3. Multiple gestation
4. Prior caesarean delivery
5. Uterine anomalies

## **Technique**

1. Sterile Ringer lactate solution or Normal saline

The fluid may be at room temperature for term gestations.

2. Double lumen intrauterine pressure catheter
3. Fetal monitor



4. Intravenous tubing
5. Continuous close monitoring using a fetal scalp electrode

### **Infusion protocols**

Many different amnioinfusion protocols have been reported.

1. 500-800ml bolus of normal saline at room temperature over 30 minutes followed by continuous infusion of approximately 3ml/minute for thick meconium stained amniotic fluid.
2. Initial bolus of 250ml normal saline at 10 to 20ml per hour infused over 20-30 minutes. An additional 250ml beyond the volume at which decelerations resolve is administered. Amnioinfusion may be repeated if gross fluid leakage occurs or variable FHR deceleration appear. The amnioinfusion is unsuccessful if infusion of 1000ml does not result in termination of deceleration.

### **Route of administration**

- a. Transcervical: Infusion of normal saline via a catheter in uterine cervix.
- b. Transabdominal performed via amniocentesis spinal needle.

This procedure is typically performed during labor through an intra uterine catheter introduced transcervically after rupture of the membranes. Alternatively fluid can be infused through a needle trans abdominally.

The transcervical route is preferred because it does not require ultrasound guidance and easily allows for repeated fluid instillation.

The fetal heart rate and resting tone are assessed continuously during the intervention.

### **Complications**

1. Uterine over distension or hyperactivity
2. Amniotic fluid embolism
3. Placental abruption
4. Cord prolapse
5. Chorioamnionitis
6. Uterine rupture
7. Maternal cardiopulmonary compromise

### **Role of amnioinusion**

The passage of thick meconium inutero puts the neonate at risk for meconium aspiration. The risk of meconium aspiration is high in patients with thick meconium particularly when it is associated with episodes of fetal hypoxemia. Thin meconium is not associated with increased perinatal mortality rate or an increased increase of meconium aspiration syndrome. Therefore any mechanism by which thick meconium can be converted to thin meconium has a positive effect on neonatal outcome and decrease the incidence of meconium aspiration.

In meconium stained amniotic fluid aminoinfusion

- a. Is used to reduce the risk of meconium aspiration syndrome by diluting the meconium.
- b. Artificially by increasing amniotic fluid volume corrects oligohydramnios resulting from rupture of membranes, thereby reducing vagal stimuli due to cord compression and thus reduce the number and severity of variable deceleration. These probably decrease further meconium passage as well as remove a stimulus for deep breathing and gasping and prevents meconium aspiration.
- c. Amnioinfusion may allow spontaneous vaginal delivery and avoid the necessity of operative intervention.

## **SIGNIFICANCE OF MECONIUM IN AMNIOTIC FLUID**

### **Meconium**

Meconium is a viscous green fluid and consists of gastrointestinal secretions, bile, mucous, desquamated fetal cells, lanugo, vernix, blood and amniotic fluid. The dark greenish black appearance is caused by pigments especially biliverdin.

### **Meconium passage**

- d. Inutero passage of meconium is uncommon due to relative lack of strong peristalsis, good anal sphincteric tone and the viscous meconium cap at the lower end of gastrointestinal tract.
- e. Three theories have been suggested to explain fetal passage of meconium which may explain the tenuous connection between the detection of meconium and infant mortality.
  - i. Inutero passage of meconium may be normal gastrointestinal tract maturation and neural control i.e, from normal bowel peristalsis.
  - ii. The pathological explanation proposes that the foetus pass meconium in response to hypoxia and that meconium therefore signals fetal compromise.

Hypoxia stimulates arginine vasopressin (AVP) release from the fetal pituitary gland. AVP stimulates the smooth muscle of colon to contract resulting in intra amniotic defaecation.

- iii. Third meconium passage could follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased peristalsis. Thus fetal release of meconium also could represent physiological process.

### ***1. The effect of maturity and gestation on passage of meconium***

Meconium is found in fetal gut from 10 wks gestation. But meconium passage is uncommon before 37 wks and most common in post term pregnancy. After 42 wks it is 30-50%, 30% at 40 wks and 50% at 42 wks. It reflects fetal gastrointestinal maturity and increased gut motility as gestational age increases.

Meconium passage in the preterm foetus can occur if it becomes infected with organisms that cause fetal enteritis (eg. listeria, ureoplasma etc).

### Cumulative meconium passage by gestational age

Study	Meconium passage
Eden and associates (1987)	
39 wks	14
40 wks	19
42 wks	26
> 42 wks	29
Usher and colleagues (1988)	
39-40 wks	15
41 wks	27
42 weeks or greater	32
Steer & Co-workers (1989)	
< 36 wks	3
36-39 wks	13
40-41 wks	19
42 weeks or greater	23

## ***2. Hormonal control of passage of meconium***

The intestinal hormone motilin has been implicated in the passage of meconium in utero. It causes contraction of smooth muscle in gut wall. The levels of motilin increase with gestation and stress on the foetus could cause release of motilin and thus passage of meconium.

## ***3. Infection and passage of meconium***

Meconium stained amniotic fluid is associated with increased peripartum infection rates especially thick meconium. It is unclear whether the presence of meconium encourages infection or whether infection promotes the passage of meconium.

## ***4. Obstetric cholestasis***

The risk of meconium passage increases in association with cholestasis of pregnancy.

## **Risk Factors**

Earlier it was suggested that fetal hypoxia causes meconium passage in utero, but it is now stated that hypoxia and acidosis per se do not lead to passage of meconium.

The predictive value of meconium as an indicator of asphyxia is better when it occurs in high risk patients and when it is dark thick and tenacious.

Passage of meconium into normal amniotic fluid results in its aspiration before labour and in healthy well oxygenated foetus this diluted meconium is cleared from lungs by normal physiological mechanism. When this meconium is not cleared, the meconium aspiration syndrome results. It can occur after normal labour but more likely in

- Post term pregnancy
- IUGR baby
- Diminished amniotic fluid volume with cord compression
- Uteroplacental insufficiency

The compromised fetus cannot clear the thick undiluted meconium.

### **Meconium aspiration**

The passage of meconium is not a risk to the foetus, but aspiration of meconium into the fetal lung is associated with clinical disease ranging from mild respiratory distress to severe respiratory compromise and occurs in 1/3rd cases. Meconium aspiration is defined as the presence of meconium below vocal cords and occurs in 10-40% of live births with meconium stained amniotic fluid.

Meconium aspiration may occur

- Inutero
- During the process of birth or



- After birth

It seems likely that meconium aspiration is largely an intrauterine event. Two types of breathing movement cause inhalation of amniotic fluid in utero gasping and deep breathing. Gasping usually occurs during hypoxemia. But in majority of infants the exact timing is not clear.

Postnatal inhalation can occur late in the second stage or immediately after delivery if the infant gasps or makes breathing movement while the oropharynx, hyopharynx or trachea contains meconium stained amniotic fluid.

## **MECONIUM ASPIRATION SYNDROME**

Meconium aspiration syndrome represents a wide spectrum of disease from transient respiratory distress with little therapy required to severe respiratory compromise requiring prolonged mechanical ventilation and high levels of oxygen administration.

It is the commonest cause of respiratory distress in term and postterm infants and its severity linked to coexisting fetal asphyxia and ultimately leads to respiratory failure. meconium aspiration syndrome occur in 10-40% of infants both through meconium stained amniotic fluid.

Occurs in more than 30% of pregnancies that continue past 42 wks.

Of infant delivered through MSAS 5-33% develop respiratory symptoms and radiographic changes of meconium aspiration syndrome.

Up to 30% of these infants require mechanical ventilation.

Approximately 1/3<sup>rd</sup> develop persistent pulmonary hypertension which contributes to the mortality associated with the syndrome.

### **Pathophysiology of meconium aspiration syndrome**

The pathophysiology of meconium aspiration syndrome is extremely complex due to interplay of number of mechanisms.

The respiratory disorders occurs of

- a. Obstructive nature of the aspirated viscous meconium which produces airway obstruction with obstructive emphysema due to ball valve effect.
- b. Pronounced inflammatory response occurs within hours of aspiration and protein leaks in the airways occur.
- c. Cytokines and eicosanoids are often present and may contribute to pulmonary vasoconstriction and tissue damage and persistent pulmonary hypertension.
- d. Meconium causes dysfunction of endogenous surfactant.

- e. Meconium affects neutrophil function, and is associated with increased risk of infection.

### **Clinical features**

After resuscitation, initial apnoea is followed by progressive respiratory distress with intercostal retraction and grunting. The AP diameter of the chest increases and appears emphysematous. The course is progressive during initial 48-72 hrs and may be complicated by persistent pulmonary hypertension leading to intractable hypoxemia and acidosis.

X-ray findings range from classic description of diffuse patchy, infiltrates or lobar consolidation to a relatively clear virtually normal appearance of lung fields.

### **Prognosis**

The outcome of meconium aspiration syndrome is guarded. The case fatality rate varies between 5% to 35%. There is a high risk of cerebral palsy, seizures and mental retardation among survivors depending upon the severity and duration of perinatal asphyxia. These infants are vulnerable to develop recurrent respiratory infection, reactive airway disease and chronic pulmonary insufficiency during childhood.

## **Management of infants born through meconium stained amniotic fluid**

### ***Intrapartum management***

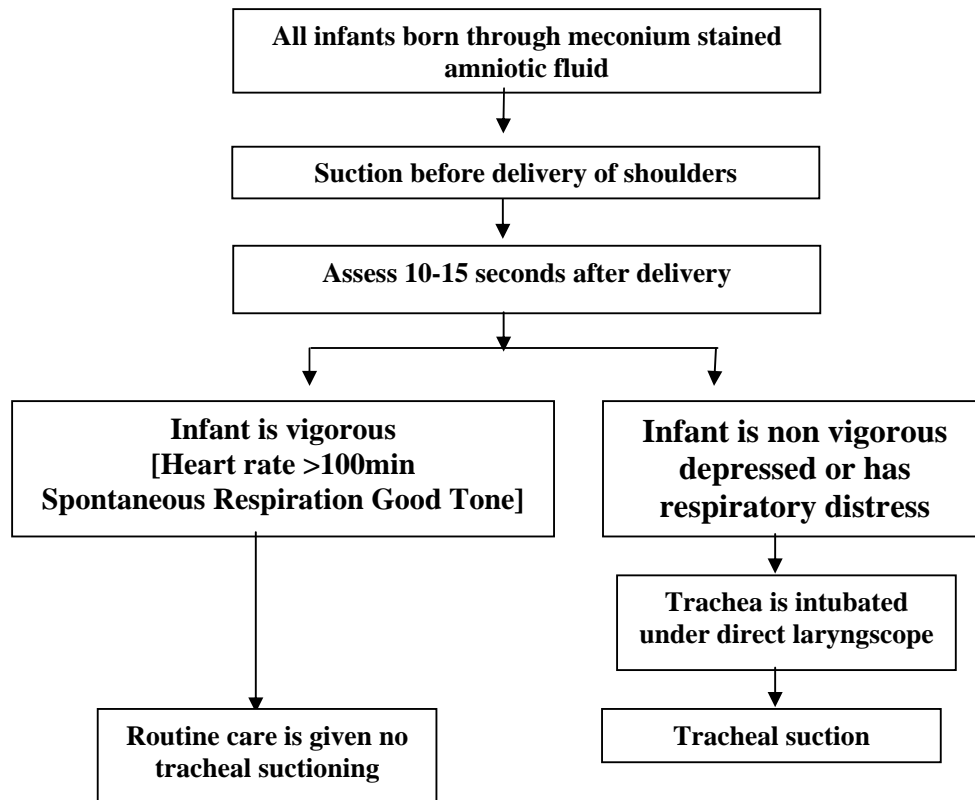
If meconium is detected continuous electronic fetal heart rate monitoring during labour is recommended. If fetal heart rate pattern is abnormal immediate delivery by caesarean section is considered.

When meconium of any consistency is present intrapartum, the obstetrician should clear the infants nose and oropharynx before delivery of shoulders with a bulb syringe or a Dee Lee suction catheter.

Recently it has been suggested that routine oropharyngeal suction in the absence of evidence of hypoxia should not be performed as it does not reduce the incidence of meconium aspiration syndrome.

### ***Postpartum management***

At delivery a pediatrician should be present (experienced in neonatal resuscitation).



20-30% infants will be depressed at perineum. In these cases the baby is handed over to the pediatrician. The trachea is intubated under direct laryngoscopy before initial respiratory efforts have been initiated and intratracheal suctioning is done to remove the meconium from the airway.

The infant's general condition should not be ignored during the attempts to clear the trachea. This procedure should be accomplished

rapidly and ventilation with oxygen should be initiated before significant bradycardia occurs.

### **Management of Meconium Aspiration Syndrome**

- Infants depressed at birth and have had meconium suctioned from trachea are at risk from meconium aspiration pneumonia and closely observed for respiratory distress.
- Oxygen saturation is monitored.
- X-ray chest is taken.

#### ***1. Routine care***

Baby should be nursed in

- Thermoneutral environment and
- Oxygen is given by head box
- Mechanical ventilation when respiratory failure is impending
- Fluid and nutritional support
- Broad spectrum antibiotics in infants with respiratory distress and abnormal radiological findings
- Corticosteroids are contraindicated
- Indwelling arterial catheter is required for blood sampling and arterial blood gas analysis

## ***2. Non conventional management includes***

- High frequency ventilation
- Exogenous surfactant
- Inhaled nitric oxide
- Liquid ventilation
- Extracorporeal membrane oxygen [ECMO]

## ***3. Acute complications***

- a. Air leak pneumothorax or pneumomediastinum occurs in 10-20% of patients with meconium aspiration syndrome more frequently with mechanical ventilation.
- b. Pulmonary hypertension associated with meconium aspiration syndrome occurs in 35% cases and inhaled nitric oxide is given.

## **MATERIALS AND METHODS**

This study was conducted in Govt. Rajaji Hospital, Madurai from Sept 2006 to Sept 2007.

200 patients with pregnancy at or beyond 37 weeks with a single live fetus in cephalic presentation and moderate to thick meconium stained amniotic fluid was included in the study.

It was a prospective comparative evaluation of two groups of 100 women one group receiving amnioinfusion for meconium stained amniotic fluid and one receiving standard care (control).

Group A - Consisted of those who received amnioinfusion

Group B - Those who did not received amnioinfusion

(Control)

All women had moderate to thick meconium stained amniotic fluid seen after either spontaneous or artificial rupture of membranes.

### **Inclusion criteria**

1. Singleton pregnancy
2. Vertex presentation
3. Gestational age of 37 wks or more
4. Normal fetal heart rate
5. Moderate and thick meconium



6. Cervical dilatation of 3-8cm
7. Women in active labour
8. No major medical or obstetric complications.

### **Exclusion criteria**

1. Major fetal anomaly
2. Chorioamnionitis
3. Placenta praevia
4. Vaginal bleeding
5. HIV infection
6. Polyhydramnios
7. Previous uterine incision
8. Multiple gestation
9. Fetal distress

### **Procedure**

After a detailed history taking including complications during the present pregnancy general physical examination and obstetric examination was made.

Gestational age was confirmed by menstrual history, fundal examination and ultrasound.

Routine haematological and urine examination was done.

All women had moderate to thick meconium stained amniotic fluid seen either after spontaneous or artificial rupture of membranes.

Further cervical dilatation, presentation, prolapse of cord, character of the meconium stained amniotic fluid and fetal heart rate variability was noted.

Thin meconium was defined as watery. Moderately meconium stained amniotic fluid as opaque without particles. Thick meconium as “pea soup” particles.

In the study group after explaining the procedure to the patient and getting her consent, under all aseptic precautions the amnioinfusion catheter (Ryles tube No.16) was inserted transvaginally and passed above the baby’s head with one end inside the uterine cavity and the other end outside connected outside to an intravenous tube. 500 ml of normal saline at room temperature was infused over 30-45 minutes until the meconium was washed out and the returning amniotic fluid was clear. In most patients 1 litre was transfused over 45 minutes.

- Intravenous broad spectrum antibiotics was given for all the patients.
- The control group did not receive any intrapartum amnioinfusion.

- All women were monitored by fetal heart sound auscultation every 15 minutes. Criteria for fetal heart rate variation was both tachycardia and bradycardia.
- Uterine activity was assessed every half an hour by palpation.
- Augmentation for inadequate uterine contraction was done with syntocinon drip when no fetal bradycardia was recorded.
- Outlet forceps or emergency caesarean section was done in those cases where there was a further deterioration of FHR, persistent bradycardia and non progress of labour.
- A paediatrician was present at the time of delivery.
- All neonates were managed by standard protocol of immediate oropharyngeal suction at delivery of the head by the obstetrician.
- The paediatrician undertook subsequent resuscitative procedures.

- During fetal oropharyngeal suction, the presence of meconium below the vocal cords on laryngoscopic examination was documented.
- If the infant was vigorous, no endotracheal intubation or suctioning was done.
- The mode of delivery, duration between detection of meconium stained amniotic fluid, amnioinfusion and delivery of the foetus was noted.
- Neonatal details considered were mode of delivery, apgar score of baby at 1 minute and 5 minutes, birth weight, presence of meconium below the vocal cords on laryngoscopic examination and meconium aspiration syndrome.
- Evidence of IUGR, post maturity and congenital anomalies were looked.
- Admission to nursery and complications including mortality were noted.
- The parameters studied included operative intervention rates, meconium below vocal cords, meconium aspiration

syndrome and post partum complication in the mother in the first 48 hrs.

- Baby and mother were carefully followed in the postnatal ward for any morbidity and mortality.

## OBSERVATION

200 patients attending Govt. Rajaji Hospital were included in this study. Group A (100 patients) : study group with amnio infusion given.

Group B (100 patients) control group – No amnio infusion

**Table 1**  
**Age Distribution**

Age in years	Study Group (A)		Control Group (B)	
	No.	%	No.	%
18-20	2	2	7	7
20-24	56	56	49	49
25-29	30	30	37	37
30-34	10	10	5	5
35 & above	2	2	2	2
Total	100	100	100	100
Mean	24.53 years		24.26 yrs	
S.D.	3.51 years		3.88 yrs	
‘p’	0.5958 Not significant			

88% in the study group and 93% in the control group were below 30 group of age. Only minority were above the age of 30 years. 12% in study group and 7% in control group. Age distribution was comparable among the two groups and majority (90%) of the patients were between 18-30 years age group.

**Table 2****Obstetric Index**

<b>Obstetric Index</b>	<b>Study Group A</b>		<b>Control Group B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
Primi	51	51	54	54
Multi	49	49	46	46
Total	100	100	100	100
‘p’	0.777 (Not significant)			

The two groups were similar in the distribution of gravidity. In Group A: 51% of the patients were primigravidas and 49% were multigravidas. In Group B: 54% of the patients were primigravidas and 46% were multigravidas.

**Table 3**  
**Gestational Age**

Gestational Age in weeks	Study Group A		Control Group B	
	No.	%	No.	%
37-39 weeks	52	52	56	56
40-41 weeks	44	44	41	41
41 weeks & above	4	4	3	3
Total	100	100	100	100
Mean	39.26		39.15	
S.D.	2.34		2.42	
‘p’	0.8226 (Not significant)			

### **Gestational age**

In group A 52% of patients came under the gestational age of 37-39 weeks, 44% were in the gestational age of 40-41 weeks, and 4% were above 41 weeks.

In group B 56% were in gestational age of 37-39, 41% were in gestational age of 40-41 weeks and 3% were above 41 weeks.



**Table 4**  
**Cervical Dilatation**

Cervical  Dilatation in cms	Study Group  A		Control Group  B	
	No.	%	No.	%
3-5 cms	62	62	56	56
6-8 cms	38	38	44	44
Total	100	100	100	100
Mean	5.45 cms		5.6 cms	
S.D.	1.78		1.92	
‘p’	0.4534 (Not significant)			

### **Cervical dilatation**

62% patient in group A and 56% patients in group B were having cervical dilation of 3-5 cm on inclusion.

**Table 5**  
**Degree of Meconium**

<b>Degree of Meconium</b>	<b>Study Group A</b>		<b>Control Group B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
Moderate	49	49	53	53
Thick	51	51	47	47
Total	100	100	100	100
‘p’	0.6713 (Not significant)			

49% patients in study group and 53% patients in control group had moderate meconium stained amniotic fluid. Whereas 51% patients in study group and 47% patients in control group had thick meconium stained amniotic fluid.

**Table 6**

**Oxytocin Augmentation**

<b>Oxytocin Augmentation</b>	<b>Study Group A</b>		<b>Control Group B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
Yes	38	38	31	31
No	62	62	69	69
‘p’	0.3721 (Not significant)			

62% of the study group and 69% of the control group had normal spontaneous labour.

Where as 38% patients in study group and 31% patients in control group required oxytocin augmentation in labour the ‘p’ value is 0.3721 which is not statistically significant.

**Table 7****Amnio infusion to delivery interval in hours**

<b>Amnio infusion to delivery interval in hours</b>	<b>Primi</b>	<b>Multi</b>	<b>Total</b>	<b>Cases</b>
1 hour and less	4	3	7	7
1.1-2 hours	20	18	38	38
2.1-3 hours	21	24	45	45
3.1-5 hours	6	4	10	10
Total	51	49	100	100
Mean	130.6 minutes		2 hrs 9 minutes	
S.D.	50.6 minutes		50 minutes	
‘p’	0.8279 (Not significant)			

The mean interval between amnioinfusion to delivery was 130 minutes in study group . It is a reflection of the time period the foetus has been surrounded by the environment containing meconium and there was enough time for amnioinfusion to have its effect .

**Table 8**

**Fetal heart rate abnormalities in Labour**

<b>FHR Abnormalities in labour</b>	<b>Study Group A</b>		<b>Control Group B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
Present	19	19	38	38
Absent	81	81	62	62
<b>‘p’</b>	<b>0.0048</b> <b>(Significant)</b>			

FHR abnormalities (tachycardia / Bradycardia) were less common in A group 19% compared to 38% in group B. Hence there were more cases of fetal distress in group B, the ‘P’ value is 0.0048 which is significant. Thus amnioinfusion significantly decreases FHR abnormalities

**Table 9****Mode of Delivery**

<b>Mode of Delivery</b>	<b>Study Group A</b>				<b>Control Group B</b>			
	<b>Primi</b>	<b>Multi</b>	<b>Total</b>	<b>%</b>	<b>Primi</b>	<b>Multi</b>	<b>Total</b>	<b>%</b>
Labour Natural	33	44	77	77	28	35	63	63
Outlet	6	3	9	9	9	6	15	15
LSCS	12	2	14	14	17	5	22	22
<b>‘p’</b>	<b>0.0449 (Significant)</b>							

Majority of the patients delivered by labour natural in both groups. 77% in study group vs 63% in the control group.

Outlet forceps was applied for 9% in study group vs 15% in control group. Caesarean section was done for 14% in study group and 22% in control group. The p value is 0.0449 which is significant. There is decreased incidence of caesarean section in the amnioinfused group thus amnioinfusion decreases incidence of caesarean section.

**Table 10****Intrapartum operative intervention**

<b>Intervention</b>	<b>Study Group A</b>				<b>Control Group B</b>			
	<b>Primi</b>	<b>Multi</b>	<b>Total</b>	<b>%</b>	<b>Primi</b>	<b>Multi</b>	<b>Total</b>	<b>%</b>
LSCS	12	2	14	14	17	5	22	22
Forceps	6	3	9	9	9	6	15	15
Total	18	5	23	23	26	11	37	37
<b>‘p’</b>	<b>0.0449 (Significant)</b>							

There is an increase in intrapartum intervention in control group. There is decreased number of caesarean section in study group compared to control group (14% vs 22%). There is also decrease number of outlet forceps in study groups compared to control group (9% vs 15%). Taking together the operative interventions in the study group and control group (23 patients in A group vs 37 patients in group B), there was decreased intervention in A group. P value (0.0449) which is significant. Hence amnioinfusion reduces the operative interventions.

**Table 11****Intervention for fetal distress**

<b>Reason</b>		<b>A</b>				<b>B</b>			
For		<b>P</b>	<b>M</b>	<b>T</b>	<b>%</b>	<b>P</b>	<b>M</b>	<b>T</b>	<b>%</b>
fetal	LSCS	9	2	11	11	16	4	20	20
distress	Forceps	5	2	7	7	6	5	11	11
	Total	14	4	18	18	22	9	31	31
p value = 0.0485									
For	LSCS	2	1	3	3	1	1	2	2
other	Forceps	1	1	2	2	3	1	4	4
reason									
	Total	3	2	5	5	4	2	6	6

There is increased intervention for fetal distress in group B 31% compared to 18% in group A. The p value is 0.0485 and is statistically significant. The incidence for LSCS for fetal distress in group B is high 20% vs 11% in group A. The incidence of outlet forceps was also 11% in group B, compared to 7% in group A, Hence amnioinfusion decreases the operative intervention for fetal distress.



**Table 12**  
**Apgar Score**

Apgar Score	Study Group (A)		Control Group (B)	
	No.	%	No.	%
<u>1 Minute</u>				
0-6	16	16	45	45
7-10	84	84	55	55
Mean	6.74		6.21	
S.D.	0.66		1.05	
‘p’	0.0001 (Significant)			
<u>5 Minutes</u>				
0-6	7	7	18	18
7-10	93	93	82	82
Mean	7.82		7.47	
S.D.	0.52		0.96	
‘p’	0.0032 (Significant)			

In the study group, the low apgar score (0-6) at 1 minute was seen in 16% neonates in study group compared to 45% in control group. The P value is (0.001) which is significant. Similarly the low apgar score at 5 minute was seen in 7% neonates in study group compared to 18% neonates in control group the p value is (0.0032) which is significant Thus amnioinfusion improves apgar score at 1 minutes and 5 minutes and improves neonatal out comes.

**Table 13**  
**Presence of Meconium below vocal cords**

<b>Group</b>	<b>Study Group A</b>		<b>Control Group B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
<u>Meconium below vocal cords</u>				
Present	13	13	41	41
Absent	87	87	59	59
<b>‘p’</b>	<b>0.0001</b> <b>(Significant)</b>			
<u>MAS</u>				
Among Present(13)	3(13)	23.1	19(41)	46.3
Among Absent(87)	1(87)	1.1	4(59)	6.8
<b>‘p’</b>	<b>0.0002</b> <b>(Significant)</b>			

Meconium below vocal cords visualized by direct laryngoscopy was seen in 13% neonates in group A compared to 41% patients in group B the p value is 0.001 which is significant.

Meconium aspiration developed in 3 out of 12 neonates in group A (23%) compared to 19 out of 41 neonates in group B (46.3%). The p value is 0.002 which is significant. Hence amnioinfusion decreases meconium below vocal cords and meconium aspiration syndrome.

**Table 14****Birth Weight**

Birth Weight  (in kg)	Study Group		Control Group	
	A		B	
	No.	%	No.	%
Less than 2	-	-	-	-
2-2.5	14	14	21	21
2.6-3	39	39	38	38
>3	47	47	41	41
Total	100	100	100	100
Mean	3 kg		2.94 kg	
S.D.	0.38 kg		0.39 kg	
‘p’	0.3605 (Not significant)			

There was no significant difference in the birth weight between two group and the mean birth weight in group A was 3 kg and group was 2.94kg.

**Table 15**  
**Admissions**

Reasons for admissions	Study Group A		Control Group B	
	No.	%	No.	%
Observation	10	10	12	12
MAS	3	3	15	15
MAS WITH HIE	1	1	8	8
Birth Asphyxia	2	2	6	6
HIE	-	-	1	1
Total Admission	16	16	42	42
Not admitted	84	84	58	58
'p'	<b>0.0002</b> <b>(Significant)</b>			

In the study group 16 neonates were admitted compared to 42 in the control group. In study group the incidence of meconium aspiration syndrome was 4 compared to 23 in the control group. In the study group most of the babies were admitted for observation only compared to the control group. The p value is 0.002 which is significant. Hence amnioinfusion results in decreased incidence of Neonatal intensive care unit admissions.

**Table 16****Perinatal Mortality and Morbidity**

<b>Parameter</b>	<b>Study cases A</b>		<b>Control cases B</b>		<b>‘p’</b>
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>	
Perinatal Mortality	1	1	8	8	<b>0.0175 (Significant)</b>
Perinatal morbidity	16	16	42	42	<b>0.0002 (Significant)</b>

Perinatal mortality in study group is 1% where in control group it is 8% p value of 0.0175 which is significant. In study group one neonate delivered by labour natural died after one week due to meconium aspiration with hypoxic ischemic encephalopathy (HIE-III).

Among control group B two neonates delivered by LSCS had meconium aspiration syndrome with HIE III and expired after 48 hrs. Among four neonates delivered by outlet forceps three neonates developed meconium aspiration with HIE III and one baby developed of meconium aspiration syndrome and died. Two neonates delivered by labour natural died of meconium aspiration syndrome. Hence amnioinfusion reduces perinatal mortality and morbidity.

**Table 17****Maternal Outcome**

<b>Maternal Outcome</b>	<b>Study cases</b>		<b>Control cases</b>	
	<b>A</b>		<b>B</b>	
	<b>No.</b>	<b>%</b>	<b>No.</b>	<b>%</b>
Uneventful	98	98	98	98
Chorioamnionitis	1	1	2	2
Uterine Hyper stimulation	1	1	-	-
'p'	0.6894 (Not significant)			

There was no significant difference in maternal infection in both groups (1% of amnioinfused vs 2% of control). Hence amnioinfusion does not increase the infection rate. There was one case of uterine hyperstimulation in group A.

**Table 18**  
**Outcome of the Study**

Out come	Study Group A		Control Group B		P Value
	No	%	No	%	
1.Caesarean section	14	14	22	22	0.0449
2. Foreps delivery	9	9	15	15	0.0449
3. FHR abnormalities	19	19	38	38	0.0048
4. Low Apgar <6 1 minutes	16	16	45	45	0.0001
5 minutes	7	7	18	18	0.0032
5. Meconium below the vocal cord	13	13	41	41	0.001
6.Meconium aspiration syndrome	4	4	23	23	0.002
7. Perinatal morbidity	16	16	42	42	0.0002
8. Perinatal mortality	1	8	1	8	0.0175

Amnioinfusion results in significant reduction in caesarean section rate (14% vs 22%), decreased incidence of FHR abnormalities (19% vs 38%) decreased incidence of low 1 min apgar score (16% vs 45%), 5 min apgar score (7% vs 18%), decreased incidence of FHR abnormalities (19% vs 38%) decreased incidence of meconium below vocal cords (13% vs 41%), decreased incidence of meconium aspiration syndrome (4% vs 23%), decreased perinatal morbidity (16% vs 42%), decreased perinatal mortality (1% vs 8%).

## **DISCUSSION**

In this study a total of 200 women with moderate and thick meconium stained amniotic fluid were studied and divided into two groups. 100 in each group.

Group A received amnioinfusion

Group B did not receive amnioinfusion and standard care was given.

The two groups were comparable with respect to age, parity duration of pregnancy, gestation, cervical dilatation and labour characteristics.

### **Mode of delivery**

#### **1. Operative deliveries**

The differences in the mode of delivery in both groups was significant  $p$  (0.0449). The incidence of caesarean section was 14% and outlet forceps 9% in group A compared to caesarean section 22% and outlet forceps 15% in group B. The total operative deliveries was 23% in group A compared 37% to group B. In the current study there is a drop in total operative deliveries in the study group (23% vs 37%).

2. There is reduced caesarean section in the study group compared to control group B (14% vs 22%).



3. There is reduced incidence of caesarean section for fetal distress in study group A compared to group B (11% vs 20%).

This coincides with the studies by Pierce et al who showed lower incidence of caesarean section in the amnioinfusion group (19.7% vs 24.3%) and his instrumental delivery was 18%. Similar observations of lower incidence of operative deliveries have been made by several authors Das et al (18% of amnioinfused vs 30% of control) and Sahu et al also reported similar decrease in caesarean section. Wenstroms (6/36 of amnioinfused vs 15/44 of control), Ilagan NB et al (6/38 of amnioinfused vs 19/40 of control) reported significant decreased caesarean section rate and decreased assisted delivery (2/38 vs 7/40). Sood et al also reported (12% of amnioinfused vs 42% of control) decreased caesarean section rate. Demeenus et al reported decreased assisted delivery. (15.5% of amnioinfused vs 25.35% of control) and decreased caesarean delivery Kosha et al also reported significant decrease in section rate (4% of amnioinfused vs 28% of control).

## **2. Fetal Heart Rate Abnormalities**

The incidence of Fetal Heart Rate abnormalities was 19% in study group and 38% in control group. The caesarean section rates for fetal distress in study group was 11% and 20% in control group. Similarly

outlet forceps for fetal distress in study group was 7% and 11% in control group.

Decreased caesarean section for fetal distress was reported by several authors. WuB et al (1991) reported significant reduction rates in caesarean section for fetal distress. Macri CJ & Schrimmer et al and (2/85 of amnioinfused vs 17/85 of control) Peurtas et al similarly reported (1/40 of amnioinfused vs 8/45 of control) reduction rates in caesarean section for fetal distress.

Demeenus et al reported a 6.75% caesarean section rate for foetal distress in patients receiving amnioinfusion where as Sasikala et al reported 28% of women undergoing LSCS with fetal distress.

Erikson reported no significant difference in incidence of fetal distress or caesarean section for fetal distress.

### **3. Meconium below vocal cords**

Meconium below vocal cords was evident only in 13 patients receiving amnioinfusion compared to 41 patients in the non transfusion group, hence the presence of meconium below vocal cord was very much reduced by amnioinfusion (13% vs 41%) and is comparable to the studies of Sood et al (16% in amnioinfused group vs 48% in control group). Decreased incidence of meconium below vocal cards has been consistently reported by several authors Sadavosky (0% of amnioinfused

vs 29% of control ) Wenstrom (2/36 of amnioinfused vs 16/44 of control) Macri & Shcrimmer (4/85 of amnioinfused vs 33/85 of control) Cialone (2/47 of amnioinfused vs 36/58 of control) Erikson (1/65 of amnioinfused vs 8/59 of control) Peurtas (4/40 of amnioinfused vs 14/45 of control) Pierce et al (4.9% of amnioinfused vs 22.9% of control) Shah (22% of amnioinfused vs 56% of control) Cramp 1 (4/162 of amnioinfused vs 6/163 of control) Spong et al showed no difference in both amnioinfused and control groups.

#### **4. Meconium aspiration syndrome**

The incidence of meconium aspiration syndrome was 4% in the study group compared to 23% in the non transfused group. Hence there is a decreased incidence of meconium aspiration syndrome in the amnioinfusion group. Similarly Das et al reported 4% in amnioinfused group and 18% in the control group. The decreased incidence of mecorium aspiration syndrome was reported by several authors.

Puertas (6.4% of amnioinfused vs 24.9% of control) Wenstroms (0/36 of amnioinfused vs 3/44 of control) Macri & Shcrimmer (0/85 of amnioinfused vs 5/85 of control) Cialone (1/47 of amnioinfused vs 8/58 of control) Erikson (0/65 of amnioinfused vs 2/59 of control), Mahomed (CRAMP):2 (3.1% of amnioinfused vs 12.8% of control), Pierce et al

(2.5% of amnioinfused vs 8.5% of control), Sood (6% of amnioinfused vs 20% of control).

- In the study by Sadavosky, no infants developed meconium aspiration syndrome in the study and control group.
- Kosha et al reported that neonatal parameters showed no significant difference in the amnioinfusion group.

## **5. Apgar score**

The low apgar score at 1 minute in the study group was 16% and in control it was 45%. Significantly fewer 1 minute up apgar scores <7, was reported by several author. This coincides with the study by Das et al (12% of amnioinfused vs 47% of control) Wenstrom (11/36 of amnioinfused vs 23/44 of control) and Ilagan et al (6/38 of amnioinfused vs 13/40 of control) also reported low 1 minutes apgar scores.

The low apgar score in 5min was 7% in study group vs 18% in the control group. Mohamed et al in (CRAMP) reported (9/324 of amnioinfused vs 27/336 of control). Das et al (4% of amnioinfused vs 23% of control), Shah (6% of amnioinfused vs 38% of control).

Rathore et al had only 1% babies in the study group with apgar <7 at 5 min. Peurtas et al did not have any neonate at apgar <7 at 5 minutes in his study.

## **6. Perinatal morbidity**

In this study 42 neonates in group A and 16 neonates in group B was admitted in NICU (Neonatal intensive care unit). CRAMP study reported less NICU admission (41/321 of amnioinfused vs 76/332 of control)

Rathor et al reported fewer admission to NICU compared to controls Hofmeyr in Cochrane review also reported reduction in the intensive care unit admissions.

In this study nine neonates had hypoxic ischaemic encephalopathy in group B compared to one neonate in group A. Hence a decrease in HIE in amnioinfusion group. CRAMP study also reported decreased incidence of hypoxic encephalopathy (1/320 of amnioinfused vs 14/329 of control). Hofmeyr in Cochrane data base systematic view 2000 reported decreased incidence of neonatal hypoxic ischaemic encephalopathy in the amnioinfused group.

## **7. Perinatal mortality**

In this study there is a trend towards reduced perinatal mortality in amnioinfusion group 1% compared to control group 8% (1% vs 8%). Das et al reported similar perinatal mortality rate of 1% in study group and 8.4% in control group. Shah et al (6% of amnioinfused vs 14% of control) Partha et al (1% of amnioinfused vs 3% of control). In cramp 2 study

(1.2% of amnioinfused vs 3.6% of control). The Cochrane review showed a trend towards reduced perinatal mortality in the amnioinfusion group.

#### **8) Maternal outcome**

There was no difference in maternal infection in both groups and hence amnioinfusion does not increase the rate of maternal infection.

- Pierce et al reported that amnioinfusion does not increase the rate of post partum endometritis.
- Mahomed (CRAMP) – reported that no complication related to amnioinfusion were observed.
- Usta et al reported higher incidence of endometritis
- Increased risk of chorio amnionitis – endometritis was reported by Spong et al.

## SUMMARY

200 patients admitted in the labour ward at Govt. Rajaji Hospital with moderate and thick meconium stained amniotic fluid, who met the criteria laid under inclusion categories were taken for this prospective comparative study.

They were allocated to two groups of 100 each. Group A 100 pt receiving trans cervical intrapartum amnioinfusion and Group B served as control

In each group, the mode of delivery, development of fetal heart rate abnormalities , neonatal outcome and maternal outcome were recorded.

The results of the study are tabulated, analysed and summarised as follows.

1. Majority (90%) of cases in both groups (88%) in study group and 93% in control group were between 18-30 yrs.
2. About half of the population in the study group were primigravida and the other half were multigravida Primi to multi ratio in group A being 51:49 Group B 54:46.
3. Gestational age : About 52% patients in study group and 56% patients in control group were between 37-39 wks and rest were above 40 wks.

4. More than half of the cases in this series had spontaneous labour (62% in study group and 69% in control) and the rest had augmented labour. (38% in study group & 31% in control group).
5. 62% patient in study group and 56% patients in control group had a cervical dilatation of 3-5 cm on inclusion.
6. The mean interval between amnioinfusion to delivery was 130 minutes in the study, hence there was sufficient time for amnioinfusion to have its effect.
7. About 49% patients in study group and 53% in control group had moderate meconium stained amniotic fluid where as 51% patients in study group and 47% patients in control group had thick meconium stained amniotic fluid.
8. Fetal heart rate abnormalities occurred in 19% in the study group and 38% cases in the control group. The p value is  $< 0.0048$  which is significant.
9. Mode of delivery.
  - a. In the amnioinfusion group (77%) had a vaginal delivery as compared to only 63% of control group patients.
  - b. The incidence of caesarean section was 14% vs 22% in the study and control group. Analyzing primigravida and multigravida separately caesarean section for fetal distress was required in 9% of



primigravida in the study group and 16% in the control group and in 2% and 4% of multigravida respectively.

The incidence of forceps deliveries was 9% versus 15% in the study and control group and for 7% in the study group and 11% in the control group the indication was fetal distress and the increased incidence in control group was due to fetal distress.

## **7. Neonatal out come**

- a. Low apgar score in one minute is 16% in study group vs 45% in control group, hence it is more common in group B. P value 0.001 which is significant.
- b. Meconium below vocal cords was evident in 13% patients in receiving amnioinfusion compared to 41% in the control group of patients (p value is 0.001) which is significant.
- c. Meconium aspiration syndrome was seen to be markedly reduced by amnioinfusion. It occurred in 4% cases in amnioinfused group and 23% of cases in the control group. The p value is 0.0002 which is significant.
- d. The mean birth weight in amnioinfusion group was 3kg and was comparable to control group 2.94 kg.

- e. The perinatal morbidity in amnioinfusion group was 16% compared to 42% in the control group the p value is 0.002 which is significant.
- f. The duration of nursery stay in both groups were similar.
- g. Perinatal mortality in the infusion group was 1% as compared to 8% in the study group value is 0.0175 which is significant.

## CONCLUSION

Intra partum Amnioinfusion in moderate and thick meconium stained amniotic fluid resulted in

- 1) Reduced incidence of FHR abnormalities (19%) compared to 38% who did not receive amnioinfusion.
- 2) Reduction in caesarean section rate 14% of amnioinfused vs 22% of control.
- 3) Significant reduction in intrapartum operative interventions for fetal distress 18% of amnioinfused vs 31% of control.
- 4) Significant reduction in the presence of meconium below vocal cords 13% of amnioinfused vs 41% of control.
- 5) Significant reduction in meconium aspiration syndrome 4% of amnioinfused vs 23% of control.
- 6) Significant improvement in low 1 minute apgar and 5 minute apgar scores 16% of amnioinfused vs 45% of control and 7% of amnioinfused vs 18% of control.
- 7) Significant reduction in perinatal morbidity 16% of amnioinfused vs 42% of control
- 8) Significant reduction in perinatal mortality 1% of amnioinfused vs 8% of control .

Intrapartum transcervical amnioinfusion is a safe simple and inexpensive technique that

- a. Reduces fetal heart rate abnormalities.
- b. Reduces operative interventions for fetal distress
- c. Decreases the rate of caesarean delivery
- d. Improves neonatal outcomes by preventing meconium aspiration syndrome.
- e. Does not increase the rate of post partum endometritis
- f. Decreases perinatal mortality & morbidity.

The presence of thick meconium is associated with increased perinatal morbidity and mortality. Meconium aspiration syndrome is a significant cause of perinatal mortality which can be reduced by amnioinfusion. In developing countries with limited intrapartum facilities amnioinfusion for thick meconium stained amniotic fluid improves perinatal outcome and decreases the caesarean section rates.

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# PROFORMA

**Case No :**

**Control No:**

**Name :**

**Date of Admission:**

**Age :**

**Unit :**

**Address :**

**IP No:**

**Urban : Rural**

**Type of cases :** Booked / unbooked

**LMP : EDD:**

**Menstrual H/o:** Regular / Irregular

**Period of gestation:**

**Obstetric code :** Gravida Para live Abortion

## **Inclusion criteria**

1. Singleton pregnancy
2. Vertex presentation
3. Gestational age of 37 wks or more
4. Normal foetal heart rate
5. Moderate and thick meconium
6. Cervical dilatation of 3-8cm
7. Women in active labour
8. No major medical or obstetric complications

## **Exclusion criteria**

1. Major foetal anomalies
2. Chorioamnionitis
3. Placenta praevia
4. Vaginal bleeding
5. HIV infection
6. Polyhydramnios

7. Previous uterine incision
8. Multiple gestation
9. Foetal distress

## **History**

### **Relevant past H/o**

### **General Examination**

1. Height :                      Weight :                      Temperature:

Anaemia :                      Pedal edema:

Pulse :                      BP :

CVS :                      RS :

### **Obstetric Examination**

P/A                      -                      FHR variation

P/V    →    1) Effacement

                    2) Dilatation

                    3) Station of vertex

                    4) Degree of meconium                      Moderate:                      Thick:

### **Time of Amnioinfusion :**

**Oxytocin Augmentation :** yes / no

**Foetal distress in labour :** Yes / No

### **Mode of delivery**

LN:                      OUTLET :                      LSCS:

Time of delivery:

Indication for LSCS / Instrumental delivery:

Rupture of membranes to delivery interval:

**Baby Outcome:**

1. Weight :
2. Sex :
3. Live / Asphyxiated
4. Apgar 1”  
5”
5. Meconium below cords:
6. Any Anomalies
7. Baby admitted :
8. Reason for admission
  1. Observation
  2. Asphyxia
  3. HIE (Hypoxic Ischemic Encephalopathy)
  4. MAS (Meconium Aspiration Syndrome)

**Maternal complication :** 1. Yes

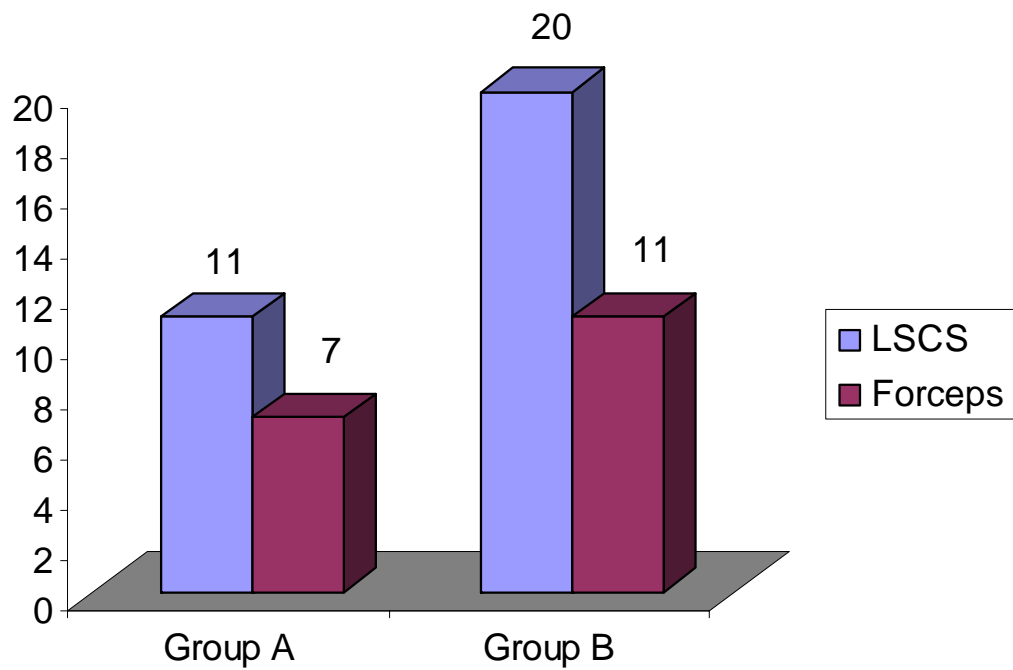
2. No

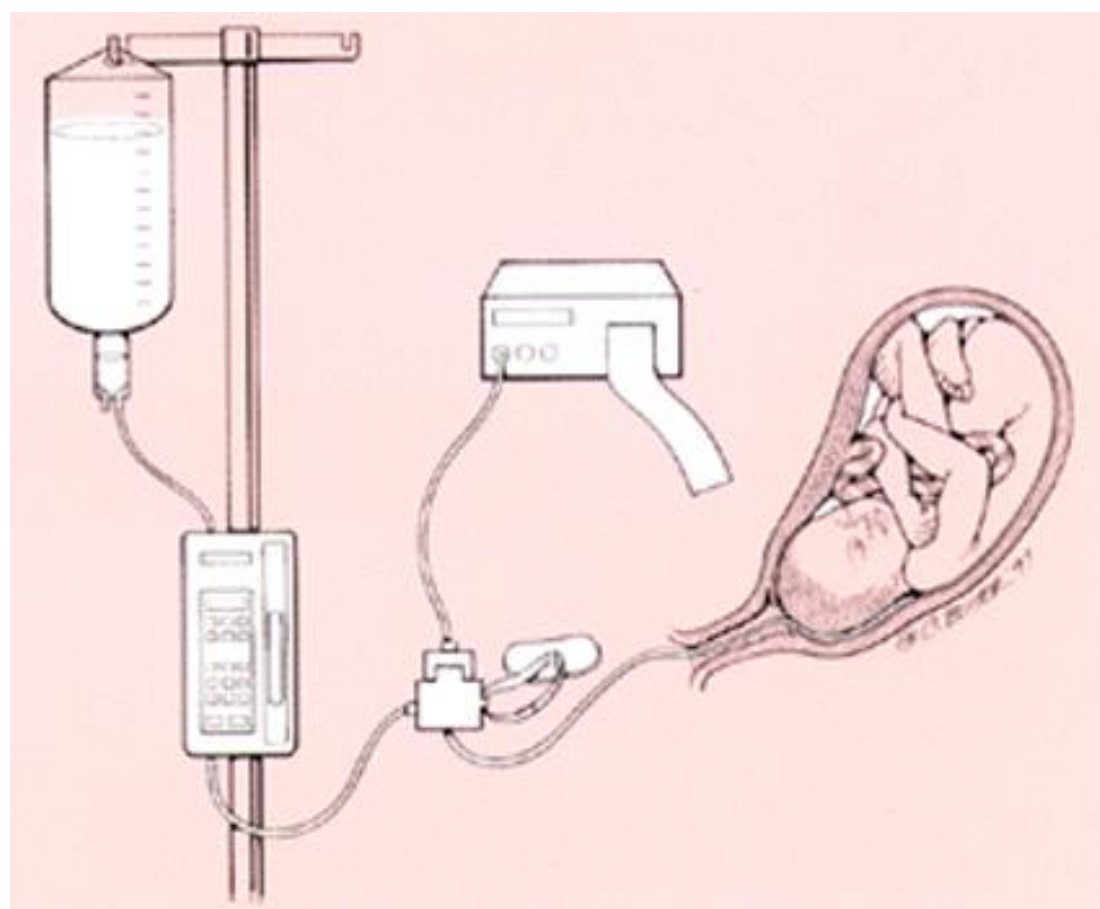
## **MASTER CHART**

### **ABBREVIATIONS USED**

AI	-	Amnioinfusion
FHR	-	Fetal Heart Rate
LN	-	Labour Natural
LSCS	-	Lower Segment Caesarean Section
FD	-	Fetal Distress
V.C.	-	Vocal cords
HIE	-	Hypoxic ischemic Encephalopathy
MAS	-	Meconium Aspiration Syndrome
B.A	-	Birth Asphyxia
Ob	-	Observation

## Intervention for Foetal distress



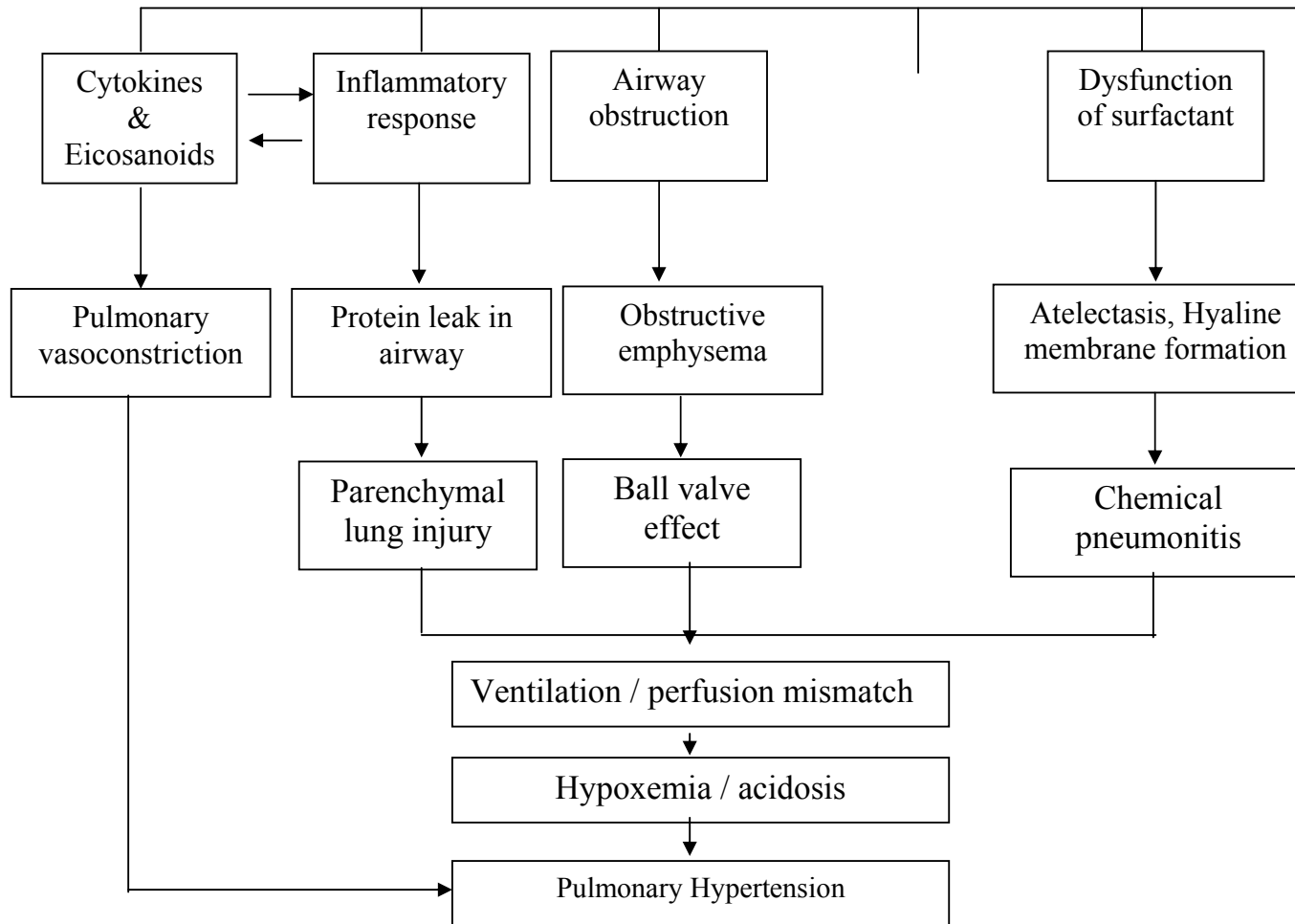






# PATHOPHYSIOLOGY OF MAS

## Aspiration of Meconium



## MASTER CHART - GROUP B

S.No. Group B	IP No.	Age	Parity	Degree of Meconium	Oxytocin Augmentation	Mode of delivery	LSCS Ind	FHR Abnormalities	AI Delivery interval	Sex	Birth Weight	Apgar 1	Apgar 5	Meconium below VC	Admission	Maternal Complication	Perinatal Mortality
1	54438	24	Primi	M	Yes	LN	-	Yes		Female	2.15	6	8	No	BA	No	Alive
2	54191	26	Multi	M	Yes	LN	-	No		Male	2.3	7	8	No	No	No	Alive
3	55803	23	Multi	T	No	LN	-	No		Female	3.1	5	8	Yes	Ob	No	Alive
4	56001	23	Primi	T	Yes	LN	-	No		Male	2.5	6	8	No	BA	No	Alive
5	54518	23	Primi	T	Yes	LN	-	No		Male	3	7	8	No	No	No	Alive
6	54529	29	Multi	T	No	Outlet	FD	Yes		Male	3.75	4	5	Yes	MAS, HIE	No	Expired
7	56446	20	Primi	M	Yes	LN	-	No		Male	3.3	7	8	No	No	No	Alive
8	55761	27	Multi	T	Yes	Outlet	FD	Yes		Female	2.8	6	8	No	BA	No	Alive
9	56107	21	Primi	M	Yes	LN	-	Yes		Female	2.5	5	6	Yes	MAS, HIE	No	Alive
10	51125	27	Primi	T	No	LSCS	FD	Yes		Female	2	7	8	No	No	No	Alive
11	54665	29	Primi	T	No	LSCS	FD	Yes		Male	3	7	8	No	No	No	Alive
12	58334	30	Multi	M	No	LN	-	No		Female	2.4	5	7	Yes	MAS	No	Alive
13	59718	24	Primi	M	Yes	LN	-	Yes		Female	2.75	6	8	Yes	Obs	No	Alive
14	58825	24	Multi	T	No	LN	-	No		Male	2.5	7	8	No	No	No	Alive
15	59940	37	Multi	T	No	LN	-	No		Male	3.05	7	8	No	No	No	Alive
16	51124	28	Multi	T	No	LN	-	No		Female	3.4	6	8	Yes	MAS	No	Alive
17	60119	23	Primi	T	No	Outlet	FD	Yes		Female	3	5	7	Yes	Obs	No	Alive
18	54995	28	Multi	M	No	LN	-	No		Female	3.1	7	8	No	No	No	Alive
19	60827	23	Multi	M	Yes	Outlet	FD	Yes		Female	3.5	7	8	No	No	No	Alive
20	62073	20	Primi	M	Yes	LN	-	Yes		Male	3.3	6	8	Yes	No	No	Alive
21	62074	30	Multi	T	No	LSCS	FD	Yes		Male	3.2	7	8	No	No	No	Alive
22	62049	19	Primi	T	Yes	LSCS	FD	Yes		Female	3.5	4.5	6	Yes	MAS	No	Alive

23	62863	21	Primi	M	Yes	LN	-	No		Female	3	7	8	No	No	No	Alive
24	63137	25	Primi	M	Yes	LSCS	FD	Yes		Male	3.3	7	8	Yes	No	No	Alive
25	64494	24	Multi	T	Yes	LN	-	No		Female	3	7	8	No	No	No	Alive
26	72284	26	Primi	T	No	LSCS	FD	Yes		Male	3.4	6	8	Yes	MAS	No	Alive
27	66072	25	Multi	T	No	Outlet		No		Female	3	5	6	Yes	BA	No	Alive
28	63442	28	Multi	T	No	LN	-	Yes		Male	2.7	4	6	Yes	Obs	No	Alive
29	63374	20	Primi	M	No	LN	-	No		Female	3.3	7	8	No	No	No	Alive
30	63427	21	Primi	M	Yes	Outlet	FD	Yes		Male	3.8	7	8	Yes	No	No	Alive
31	71996	23	Multi	M	No	LN	-	No		Male	2.6	7	8	No	No	No	Alive
32	63604	20	Multi	T	No	LSCS	CPD	No		Female	2.6	7	8	No	No	No	Alive
33	67297	26	Primi	T	Yes	Outlet	-	No		Male	3.25	5	7	Yes	Obs	No	Alive
34	63541	20	Primi	M	No	LN	-	No		Female	2.1	7	8	No	No	No	Alive
35	67372	22	Primi	T	No	LSCS	FD	Yes		Female	3.1	4	6	Yes	Obs	No	Alive
36	64342	24	Multi	M	Yes	LN	-	No		Male	3.4	5	6	No	BA	No	Alive
37	68466	25	Multi	M	No	Outlet	FD	Yes		Female	3.25	7	8	No	No	No	Alive
38	62240	18	Primi	M	Yes	LN	-	No		Male	2.8	7	8	No	MAS	No	Alive
39	63782	19	Primi	T	No	LSCS	FD	Yes		Male	3.5	7	8	No	MAS	No	Alive
40	63936	30	Primi	M	Yes	LN	-	No		Female	3.2	7	8	No	No	No	Alive
41	64030	25	Primi	T	No	Outlet	FD	Yes		Male	2.3	4	5	Yes	MAS	No	Expired
42	69803	23	Primi	M	No	LN	-	No		Male	3	5	7	Yes	No	No	Alive
43	63990	26	Multi	T	No	LSCS	FD	Yes		Female	3.25	5	6	Yes	MAS	No	Alive
44	69862	21	Primi	T	Yes	Outlet	FD	Yes		Female	3.4	5	6	Yes	Obs	No	Alive
45	64900	21	Primi	M	No	LN	-	No		Male	2.5	7	8	No	No	No	Yes
46	62211	26	Primi	M	No	LN	-	No		Male	3	7	8	No	No	No	Alive
47	63649	25	Primi	M	No	LN	-	No		Male	2.5	6	7	Yes	MAS	No	Alive
48	64055	24	Primi	T	Yes	LN	-	No		Female	2.7	6	7	No	BA	No	Alive
49	70768	23	Primi	M	Yes	LSCS	FD	Yes		Female	2.5	4	5	Yes	MAS, HIE	No	Expired
50	64230	27	Multi	M	No	LN	-	No		Male	3.5	5	7	Yes	MAS	No	Alive
51	71315	21	Primi	T	No	LSCS	FD	Yes		Female	2.6	7	8	No	MAS	No	Alive

52	71837	29	Primi	M	No	Outlet	FD	Yes		Male	2.3	6	8	Yes	Obs	No	Alive
53	71607	29	Primi	T	No	LSCS	FD	Yes		Female	3.5	6	8	Yes	No	No	Alive
54	71529	39	Multi	M	No	LN	-	No		Male	2.6	7	8	No	No	No	Alive
55	70844	21	Primi	M	No	LSCS	FD	Yes		Male	3.3	7	8	No	No	No	Alive
56	72339	19	Primi	M	No	LN	-	No		Female	3	6	8	Yes	MAS	No	Alive
57	72415	29	Primi	T	No	LSCS	FD	Yes		Male	2.6	7	8	No	No	No	Alive
58	72671	24	Multi	M	Yes	LN	-	No		Male	3	5	7	Yes	Obs	No	Alive
59	72991	19	Multi	T	No	LN	-	No		Male	2.5	7	8	No	No	No	Alive
60	76065	20	Multi	M	No	LN	-	No		Male	3.1	6	8	Yes	Obs	No	Alive
61	72818	18	Multi	M	No	LN	-	No		Female	2.2	7	8	No	No	No	Alive
62	64219	20	Primi	T	Yes	LN	-	No		Female	2.75	7	8	No	No	Yes	Alive
63	80841	27	Primi	M	No	LN	-	No		Female	3.4	7	8	No	No	No	Alive
64	78395	22	Multi	M	Yes	LN	-	No		Female	2.75	7	8	No	No	No	Alive
65	76251	25	Primi	T	No	LSCS	CPD	No		Male	2.75	6	8	Yes	No	No	Alive
66	73852	18	Primi	M	No	LN	-	No		Male	2.9	4	5	Yes	HIEII , MAS	No	Expired
67	76241	20	Multi	M	Yes	LN	-	No		Male	3.2	7	8	No	No	No	Alive
68	75867	34	Multi	M	No	LSCS	FD	Yes		Male	2.8	7	8	No	No	No	Alive
69	73864	24	Primi	T	No	LSCS	FD	Yes		Male	2.7	6	7	Yes	Obs	No	Alive
70	73809	25	Primi	M	No	LN	-	No		Female	2.5	7	8	No	No	No	Alive
71	76874	23	Multi	M	Yes	LN	-	No		Male	2.2	6	8	Yes	MAS	No	Alive
72	74025	22	Multi	M	No	LN	-	No		Female	3.2	7	8	No	No	No	Alive
73	76250	21	Primi	T	No	LN	-	Yes		Female	3	5	6	Yes	MAS	No	Alive
74	77572	25	Multi	T	No	LSCS	FD	Yes		Male	3.7	7	8	No	No	No	Alive
75	78350	21	Primi	M	Yes	LN	-	Yes		Female	2.7	7	8	No	No	Yes	Alive
76	73933	27	Primi	M	Yes	Outlet	-	No		Female	3	7	8	No	No	No	Alive
77	74063	23	Multi	T	No	LN	-	No		Male	3.3	6	8	No	No	No	Alive
78	74207	24	Primi	T	No	LSCS	FD	Yes		Female	3	7	8	Yes	MAS	No	Alive
79	74141	21	Multi	T	Yes	LN	-	No		Male	2.5	7	8	No	No	No	Alive
80	80118	24	Multi	M	No	Outlet	FD	Yes		Male	2.7	5	6	Yes	MAS, HIE	No	Expired

81	80448	22	Multi	T	No	LN	-	No		Male	3.2	6	8	Yes	No	No	Alive
82	80806	25	Primi	T	No	Outlet	FD	Yes		Female	3	4	5	No	HIE	No	Alive
83	74432	26	Multi	M	No	LN	-	No		Female	3.2	7	8	No	No	No	Alive
84	81591	34	Multi	M	No	LN	-	No		Male	2.5	3	4	Yes	MAS, HIE	No	Expired
85	83344	21	Primi	M	No	LN	-	No		Female	3.5	7	8	No	No	No	Alive
86	83532	25	Primi	M	No	LN	-	No		Male	2.8	7	8	No	No	No	Alive
87	74514	25	Primi	T	Yes	Outlet	-	No		Female	3.1	4	6	Yes	MAS, HIE	No	Expired
88	82670	24	Multi	T	No	LN	-	No		Male	2.5	5	6	No	No	No	Alive
89	83328	22	Multi	M	No	LN	-	No		Male	3.2	7	8	No	No	No	Alive
90	80831	27	Primi	T	No	LSCS	FD	Yes		Female	3	7	8	Yes	No	No	Alive
91	80817	25	Primi	M	No	LN	-	No		Female	3.5	7	8	No	No	No	Alive
92	70172	25	Multi	M	No	LN	-	No		Male	3.1	7	8	No	No	No	Alive
93	72381	20	Primi	T	No	LSCS	FD	Yes		Female	2.5	4	6	Yes	MAS, HIE	No	Expired
94	54965	24	Primi	M	No	LN	-	No		Male	3.4	7	8	No	No	No	Alive
95	54995	28	Multi	T	No	LN	-	No		Female	3.1	7	8	No	No	No	Alive
96	60621	20	Multi	T	No	LN	-	No		Male	2.4	7	8	No	No	No	Alive
97	74211	24	Multi	M	No	LN	-	No		Male	3.25	7	8	No	No	No	Alive
98	74145	28	Multi	T	No	LN	-	No		Female	3	7	8	No	No	No	Alive
99	808115	26	Multi	M	No	LN	-	No		Female	2.7	6	7	Yes	Ob	No	Alive
100	84432	25	Multi	T	No	LN	-	No		Female	2.75	7	8	No	No	No	Alive